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EDITORIAL COMMENT

Carotid Versus Brachial Pulse Pressure in Elderly Persons*

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Studies of cardiovascular physiology have shown that, for the same mean arterial pressure, systolic blood pressure (SBP) and pulse pressure (PP) are higher in peripheral (brachial) than in central arteries (thoracic aorta, carotid arteries) (1). The difference, called SBP or PP "amplification," approximates 14 mm Hg and is observed both in normotensive and hypertensive subjects. Amplification is the consequence of the progressive reduction of diameter and increase in stiffness from the proximal to the distal arterial vessels and mostly of the modification in the transit of wave reflections (1). Amplification tends to disappear with age and reduction of heart rate. It is easy to understand that this parameter contributes "per se" to protect the heart from an increase in afterload.

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In recent years, it has been suggested that increased brachial and carotid PP are both independent and significant predictors of cardiovascular events and that central PP might be a stronger predictor than brachial PP (2). In the current issue of the *Journal*, the latter finding is shown after a series of works conducted since 1992 on the matching between the heart and large arteries by Roman et al. (3). Here the results were obtained from a cohort of normotensive and hypertensive individuals followed during 8 years without any drug treatment (4). The data were collected in a small city in the center of Italy and focused on the subject of heart failure and not directly on vascular diseases. Blood pressure was measured only in 2 circumstances: in 1995 and in 2003. The major interest of the study was performed exclusively in subjects ≥65 years (mean age 73 years), thus leading to 2 important observations. First, PP amplification tends to be physiologically reduced with age, owing to a more rapid age-induced increase of central rather than brachial PP. However, it is worth noting that, at approximately 73 years, PP amplification still approximates 10 mm Hg and thus is able to consistently modulate cardiac load. Second, heart rate remains approximately 68 beats/min. Normally, even in elderly people, PP amplification is augmented in the presence of tachycardia and reduced when heart rate is lowered. In the present cohort, there was no drug treatment, and this heart rate modulation did not seem to be efficient at rest. In contrast, when elderly hypertensive subjects are resistant to drug treatment, heart rate is usually increased and participates to some extent to maintain PP amplification and the resulting cardiac load (5).

In recent years, SBP and PP have been considered independent predictors of cardiovascular risk, with a quite similar statistical power of the 2 parameters in hypertensive subjects. In elderly individuals, PP was even more powerful than SBP, better able to evaluate the main determinants of pulsatility: ventricular ejection, arterial stiffness, and wave reflections (1,2). Finally, the site of blood pressure measurements seems the most appropriate factor, enabling simultaneous determination of the mechanism and degree of cardiovascular risk. In this context, a particular example is the arterial stiffness in subjects with advanced renal failure. Stiffness is increased together at the aortic and the upper and lower limb vascular territories. However, increased stiffness is predictive of cardiovascular events only when measured at the aortic site but not at the lower or upper limbs (6). The situation seems very easy to understand—regarding PP and the heart—in hypertensive subjects of middle age. The heart directly "sees" the thoracic aorta and not the brachial artery. It is thus expected that cardiac hypertrophy might better correlate with central rather than with brachial PP (1,2,7). Inversely, the regression of cardiac hypertrophy by drug treatment is much more linked to the reduction of central and not brachial PP (7).

Coronary circulation and PP is more complex to investigate than cardiac mass. The mechanical factor to consider is not central PP but rather end or mean diastolic blood pressure. This factor is usually studied in association with the duration of diastole (1). Both factors play an independent role, because coronary perfusion occurs exclusively during the diastolic period and is largely influenced by cardiac autocontraction.

In the presence of an unstable plaque in a patient over 50 years of age, it is important to determine which factors, independent of coronary reserve, might determine the occurrence of a coronary ischemic accident. One of the most important factors to consider is again the duration of diastole, which in percentage terms plays a more important role in coronary risk than the severity of coronary stenosis itself (1). Another hemodynamic parameter is central PP and/or distensibility or even aortic stiffness (2). However, in populations over 70 years of age, carotid arterial distensibility has never been found to be a significant cardiovascular risk factor (8,9). Thus, the possibility is raised that intrinsic arterial wall properties by themselves and not distensibility might be a consistent risk factor. In the arterial circulation,

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regions of disturbed flow, particularly those characterized by flow separation and transient vortexes, are susceptible to atherogenesis, whereas regions of undisturbed laminar flow appear protected (10). Coordinated regulation of gene expression by endothelial cells has been shown to result in different regional phenotypes that either favor or inhibit atherogenesis (10). At this point it is important to recall that atheroma by itself is composed only of soft tissue, but with age, stiff tissue due to collagen might appear and modify the local phenotype.

In conclusion, the study of Pini et al. (4) shows the superiority of carotid PP over brachial PP for the prediction of cardiovascular risk. This largely reflects the role of age on the development of atherosclerotic carotid complications in elderly individuals. It seems that, in the presence of atherosclerotic plaques, the introduction of additional risk factors, such as age and/or central PP, precipitates the evolution and favors the development of the underlying lesions.

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